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Disease spread between domestic cattle and feral pigs: improving emergency preparedness

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Abstract

Studies were undertaken to understand disease transmission in feral pig populations and the implications for an incursion of exotic foot-and-mouth disease in northern Australian cattle herds. The study site was the Fitzroy River in the Kimberley region of northwest Western Australia. *Salmonella* was chosen as the indicator organism. Although infection of feral pigs was common, infection of co-grazing cattle herds was uncommon. Based on the distribution of feral pigs and cattle, a disease spread model suggested that if FMD is controlled in cattle, then it is likely to be self-limiting in feral pigs. To eradicate an FMD incursion as quickly as possible, both feral pigs and cattle should be targeted. Study findings provide a practical guide for approaching the response to an incursion of FMD in northern Australian cattle herds and associated feral pig populations.

Executive summary

This project focused on collecting field data to describe *Salmonella* transmission in commercial cattle in northern Australia (the Kimberley region) and the risk posed by the presence of feral pigs for *Salmonella* spread to cattle. The intra-herd genetics of feral pig herds associated with commercial cattle paddocks were also determined. Based on the field information collected and a disease spread model, inferences were made for potential foot-and-mouth disease (FMD) outbreaks to enhance emergency disease preparedness plans in northern pastoral regions.

Cattle sampling was completed in May 2011 on the three main pastoral leases comprising the study site. A total of 496 cattle faecal samples were collected from 47 different cattle herds in feral pig free and feral pig infested areas. Fecal samples were cultured for *Salmonella* using standard microbiological techniques. Approximately 2% of cattle samples were culture positive for *Salmonella*, a prevalence much lower than that of co-grazing feral pigs (~38%). Additionally, cattle *Salmonella* isolates were more commonly from areas without feral pigs, but conversely, from high density cattle populations on artificial water. No associations between the serotypes identified from cattle and those identified in the feral pig population were found. Fingerprint analysis (using pulsed-field gel electrophoresis) confirmed the lack of an association.

The feral pig population in the study area was sampled and genotyping was completed. A total of 543 feral pig genotypes were analysed across 14 loci. The feral pig genetic population was found to be remarkably homogenous across the large catchment area of the Fitzroy River.

Feral pig and grazing cattle distributions were created based on aerial surveys of the study area and expert opinion. A susceptible-infected-resistant disease spread model was coded and parameterised based on published literature and expert opinion.

A baseline scenario in which infection was introduced via feral pigs, with transmission from pigs to cattle and no disease control, was simulated. Assumptions regarding disease transmission were investigated via sensitivity analyses. Predicted size and length of outbreaks were compared assuming different control strategies based on movement controls, surveillance and depopulation.

Based on field studies of the interaction between domestic cattle and feral pigs in the Kimberley region, the potential spread and control options for an FMD incursion in northern Australia were investigated, using a disease spread model. Depopulation of feral pigs only was not predicted to be successful. Movement standstill, surveillance and depopulation of cattle only would successfully eradicate the disease. However, control targeting both feral pigs and cattle would result in smaller outbreaks. If FMD is controlled in cattle, then it is likely to be self-limiting in feral pigs. To eradicate an FMD incursion as quickly as possible, both feral pigs and cattle should be targeted.

Based on the research conducted and parallel research funded the Australian Research Council, Cattle Council, Department of Agriculture, Fisheries and Forestry, and Department of Agriculture and Food Western Australia, the following recommendations are made:

1. **The immediate response to an incursion of FMD that might involve feral pigs should be carefully considered.**

   Disease may not transmit across the landscape as rapidly as previous research indicated. Based on the current research, disease is likely to spread relatively slowly (assuming no human-mediated spread). This gives those responsible for responding to such an incursion time to consider which plan should be implemented.

2. **Limited culling of feral pigs is a useful response to an FMD incursion, and in the absence of additional field information should aim for 60-80% of the population within 10-20km of the index case.**

   If feral pig culling is used to contain and eradicate disease, the required proportion and distance that needs to be culled surrounding an index case might be in the order of 60-80% for 10-20 km in most cases. This is much lower than suggested by previous research and limited culling in the environment studied to eradicate disease might be eminently feasible.

3. **Surveillance should be undertaken before any mass culling campaigns of feral pigs are initiated.**

   If feral pigs and managed cattle are co-grazing then cattle should be the focus of control – there are higher numbers and they are moved more frequently. The status of the associated feral pig population should be assessed. It is possible that they will remain uninfected – or infection may be very limited – and only a focused subsequent feral pig culling program may be required. During the earlier stages of an incursion, radial sampling can be optimal, but at later stages leapfrog sampling will outperform radial sampling.

4. **During culling of feral pigs every effort should be made to remove each mob and adjacent mobs.**

   Within feral pig populations in the study area, disease is likely to spread by local social contact and over short distances. Culling efforts should concentrate on contiguous habitat. There should be particular concentration on resource rich areas.

This research has provided information that advances our understanding of the role that feral pigs might play in an FMD incursion in northern Australia, and what is the best way to respond to such a crisis if feral pigs are thought to be involved.

Table of Contents

1. Background

Overseas, feral pigs are involved in outbreaks of foot-and-mouth disease (FMD). The costs of an outbreak of FMD to Australia has been estimated at $6−13 billion and would be very damaging for Australian red meat producers.[1] Fortunately, contemporary technologies (molecular tools and disease simulation approaches) provide a unique opportunity to assess the role of feral pigs in potential FMD incursions and to improve preparedness and response planning.

Understanding wildlife disease epidemiology is critical to managing disease risks. However, designing and implementing studies that generate high quality epidemiologic data are challenging, because of the cryptic nature of wildlife and the high conservation value of many wildlife species. Despite this, there are still excellent opportunities. For example, many species of introduced animals function as free living wildlife populations. These invasive or feral species are often at high densities and are deliberately controlled. These feral wildlife populations thus offer solutions by allowing practical collection of data essential to understanding wildlife disease epidemiology. Additional solutions are offered through the application of newer genetic and modelling techniques, which creates a powerful approach to collecting and interpreting data from such populations.

Feral pigs are an invasive species in Australia that cause agricultural, economic and environmental damage.[2] In Australia they are a reservoir host for important zoonotic (animal-to-human) diseases such as *Brucella suis* and melioidosis.[2] Overseas they transmit and act as reservoir hosts for emergency trans-boundary diseases such as classical swine fever (CSF).[3] Feral pigs or wild boar have been intimately involved in FMD outbreaks in several countries, including Israel and Turkey. They have therefore been perceived as a major biosecurity threat in Australia.[2]

Limited research has been conducted in Australia and internationally to investigate disease epidemiology in feral pigs. Usually this research has involved simple sero-surveillance (e.g. see references 4 and 5) or desk top disease modelling exercises to investigate the theoretical epidemiology of outbreaks of trans-boundary diseases in feral pigs, especially for FMD (for examples, see references 6–9). Many of these models have employed simple mass-action approaches, solved by purely analytical means. Although these methods can produce useful strategic outcomes such as the estimation of a threshold density, they are often based on unrealistic assumptions such as homogenously mixing populations.[10]

Poorly defined measures of transmission dynamics are also a critical barrier in our understanding of disease epidemiology derived from these models.[6] Important parameters, such as the transmission co-efficient, have been estimated in several non-optimal but pragmatic ways including expert opinion,[6] feral pig proximity during observational studies[7] and through mathematical analysis of an overseas outbreak.[11] Only one study has actually

used empirical data to infer transmission dynamics within Australian feral pigs,[4] but was limited by the low resolution of sero-surveillance.
Contemporary technologies (molecular tools and simulation approaches) provide new opportunities to examine and test hypotheses in wildlife disease epidemiology. Such an innovative and integrative framework is well suited for application to feral pigs: its application will enhance conceptual knowledge of wildlife transmission dynamics in general, and will specifically improve our response to outbreaks of emergency trans-boundary diseases in invasive species and other wildlife populations in Australia.

Simulation modelling approaches have undergone a revolution in recent decades[12] and can now be used to create new generation, spatially explicit epidemiological models that truly represent the complexity of wildlife disease epidemiology. These models can capture the key ecological, behavioural, spatial and temporal features of a feral pig system and hence will not be bound by many of the simplifying assumptions of other approaches.[10,13] They are also flexible enough to allow examination of various mitigation strategies for efficacy, practicality and cost effectiveness and can inform government disease preparedness policy.[14]

Despite advances in modelling techniques, the lack of field data to conceptualise or estimate key population and epidemiological parameters hampers the application of simulation modelling to real problems of disease transmission in wild species. Modern molecular ecological techniques have been used to generate population genetic data useful to infer familial and meta-population structures of feral animals and other wildlife.[15,16] Although many molecular epidemiological tools are available, they have rarely been applied to explore infectious agent epidemiology in any host population.[17] It is possible to combine host population genetic studies with the molecular epidemiology of their infectious diseases to provide a much deeper understanding of transmission dynamics within wild populations, and between wild and domestic species. For example, Siddle et al.[18] demonstrated allograft transmission, and highlighted the mechanism of graft survival by exploring microsatellite diversity and major histocompatability complex diversity at both the host and tumour level in the Tasmanian devil (*Sarcophilus harrisii*). However, in that particular case, it was not possible to explore both disease and host variability in parallel to infer transmission dynamics in the Tasmanian devil, because the “disease” was an allograft.[18] Similarly, the risk of introduction of Avian Influenza from wild birds moving between continents has been examined using both host and infectious organism genetics but at too coarse a scale to infer disease dynamics directly. Thus, an approach combining the genetic analysis of both host and disease at an appropriate scale of sampling is yet to be examined for characterising disease dynamics in wild populations.

Here, we use an innovative approach integrating field and laboratory epidemiological analyses, simulation modelling, modern population genetic techniques and analysis of demographic and environmental data to fully examine, for the first time, the role that feral pigs might play as reservoirs of trans-boundary emergency animal diseases – such as FMD – in the event of a

disease incursion. The output of such research is designed to enhance contingency plans for emergency animal diseases, thus protecting Australian livestock industries and the national economy.

1.1 Purpose and description

The Researchers focussed on collecting field data to describe *Salmonella* transmission in commercial cattle in northern Australia (the Kimberley region). The risk posed by the presence of feral pigs for *Salmonella* spread to cattle was estimated and inferences made to potential FMD outbreaks. The researchers tested whether presence of feral pigs affected salmonellosis in cattle and whether having homogenous, highly related herds of feral pigs (that may be inferred to be highly mobile) were a risk factor for a diverse *Salmonella* microflora in cattle herds.

Data were collected from both feral pig infested and pig free areas. The researchers also determined the intra-herd genetics of those feral pigs herds associated with commercial cattle paddocks. Using these data, parameters were derived to inform FMD simulation models and modelling was conducted to enhance emergency disease preparedness plans in northern pastoral regions.

2. Project objective

To understand whether the presence of feral pigs and feral pig herd structure is a risk factor for salmonellosis or salmonella diversity in sympatric cattle. This will allow parameterisation of simulation models for FMD, and thus improve preparedness and response plans for potential exotic disease incursions, to protect Australian grazing industries and the national economy.

2.1 Project aim

1. Understand disease transmission between northern commercial cattle and feral pigs.
2. Evaluate key mitigation strategies for managing exotic diseases.

2.2 Project outcome (Deliverables)

1. Quantified measures of disease dynamics between feral pigs and cattle
2. Parameterise a disease simulation model for FMD based on a cattle production area
3. Improvements to preparedness and response plans for exotic disease (e.g. enhanced surveillance and mitigation strategies)

2.3 Project milestones

1. Cattle sampling plans complete (e.g. travel bookings, animal ethics committee approval, purchase of laboratory consumables etc) [30 April 2011]
2. Feral pig intra-herd genotyping complete [30 August 2011]
3. Cattle sampling complete [31 December 2011]
4. Cattle Salmonella analyses completed [1 July 2012]
5. Quantified measures of disease dynamics complete, disease spread simulations run [28 February 2013]
6. Recommendations for changes to AusVetPlan made [1 July 2013]

3. Methodology

Cattle sampling and analysis

Although cattle were kept in separate paddocks, albeit some very large, and thus may constitute separate sub-populations, the population of interest was considered cattle sharing a habitat with feral pigs and so was considered as a whole for the purposes of this calculation.

Sample size calculations were performed using Win EpiScope 2.0 sample size calculation estimating percentages function and assuming simple random sampling. A prevalence of 8% was assumed, with 5% precision, 95% level of confidence and an estimated population size of 10,000 estimates. The sample size was inflated to 600 to allow for sample transportation issues.

Meetings with collaborating leaseholders in the Fitzroy Crossing district were undertaken during the second week of May 2011. Sampling was undertaken during May/June 2011.

Faecal samples were obtained from mobs of cattle. Areas of grazing cattle where there were high feral pig densities, and low feral pig densities, were targeted.

For efficient sampling, samples were collected via the use of helicopters. Paddocks to be sampled were targeted and flown using an Robinson R44 helicopter. When the first mob of cattle was spotted, they were observed for 1-2 minutes, the helicopter circling the mob. Then on landing, fresh faecal samples were collected from individual pats. Ten 5ml samples were collected per site from the centre of each individual dung pat. Care was taken to ensure that the dung pat was fresh.

Samples were immediately stored at 4^{o}C. Samples were transported for *Salmonella* culture to the Elizabeth MacArthur Agricultural Institute, Menangle, NSW, within 72 hours of collection.

Isolation of *Salmonella* from faecal samples was attempted using standard bacterial culture techniques based on Australian standards (AS 5013.10-2009

Microbiology of food and animal feeding stuffs – Horizontal method for the detection of *Salmonella* spp.).

Salmonella selective broths, mannitol selenite broth (MSB) and Rappaport–Vassiliadis broth (RV) were inoculated with one gram of faeces. MSB and RV broths were incubated overnight at 37^{o}C and 42^{o}C, respectively. Broths were streaked for isolation on XLD Agar plates that were incubated at 37^{o}C overnight. For presumptive testing, up to three suspect *Salmonella*-like colonies were sub-cultured onto individual 5% Sheep Blood Agar (SBA) plates and incubated at 37^{o}C overnight. Presumptive screening of Salmonella-like colonies was performed using a *Salmonella* latex agglutination test (OXOID DR1108A). Further biochemical testing, consistent with *Salmonella* species likely to be found in warm blooded terrestrial animals was performed on all positive latex cultures using Triple Sugar Iron (TSI) Agar slope and ß-galactosidase tests. Isolates producing results consistent with *Salmonella* were subcultured onto Nutrient Agar slopes and submitted for serotyping to the Australian *Salmonella* reference laboratory (www.imvs.sa.gov.au). All *Salmonella* isolates were stored on Protec Beads (OXOID Australia, Catalogue Number TS/70) at -20^{o}C.

Feral pig sampling and analysis

Feral pigs were spotted by an observer in a Robinson R44 helicopter. All permanent water sources in the known distribution of feral pigs in the study area[19] were searched for pigs. Following the cull of 10–50 pigs during a flight, a sampling team was then flown to the site(s) where pigs had been culled and measurements were made and samples were collected. Sampling was usually achieved within one hour of culling. All pigs discovered at each location were culled and sampled. Sampling was undertaken between 18 and 27 October 2010. Each sampling location was identified using a hand-held GPS. For genetic analysis, skin (approximately 2 x 0.5cm square tissue samples were taken from the least hairy part of the pinna), and from pregnant sows, fetal genetic samples (2 x nose or tail tissue samples from 2 foetuses, when possible) were collected.

Tissue samples collected for genetic analysis were analysed across 14 loci to characterize pig genotypes within the sampled population. DNA was extracted from pig ear tissues using the Machery Nagel NucleoSpin Tissue kit. Fourteen pig microsatellite markers[20,21] were amplified from DNA samples in three multiplex PCRs using the Qiagen Multiplex PCR kit. Microsatellites were genotyped on a Beckman CEQ8000 and alleles were scored using the CEQ 8000 Genetic Analysis System software Version 8.0.

Sampling was approved by the University of Sydney Animal Ethics Committee project approval N00/6-2010/5319.

Disease spread modelling

An aerial survey was conducted (designed using Distance 6.0 [22] automated algorithms and distance sampling[23] and mark-resight (double observer)[24]

methodology) to estimate the abundance and distribution of feral pigs and to assess group sizes of cattle, and results were combined with previous population estimates for cattle and pigs[25-27] and leaseholder information to develop population distributions for disease spread modelling.

Transects were flown in a North–South direction at 50 m height above ground level and a speed of ~45 kph in an R44 helicopter. Surveys were undertaken in August 2010 in the first 3 hours after sunrise (approximately 6:00 am) and the last 3 hours before sunset (approximately 5:23 pm). Areas around major permanent water sources (the Fitzroy and Margaret rivers) were searched for up to 4km to include likely feral pig home ranges.[2,10,28] The study area was searched using systematic random transects to cover areas of expected high and low density.[29,30]

Animals were enumerated by subitised recordings using a specially modified keyboard linked to a portable notebook laptop running a specialist aerial survey recording software,[a] which includes a continuous GPS track and sighting recording system.[31] Sighting distances were based on vertical sighting poles attached to a cross beam fixed through the cabin of the helicopter. Transect sightings data, reconciled for the three observers, was imported into Distance 6.0.[22] Data were analysed using distance methods, as recommended by Buckland et al.[29] and mark recapture (double observer) techniques,[32] using the DISTANCE software.

The average cattle density in the west Kimberley region is reported to be to be 7 cattle per sq. km.[25] For modelling purposes, a spatial data set of cattle herds was synthesised. A herd is defined as a co-mingling group of grazing cattle which can be considered the basic epidemiological unit for disease transmission purposes in this environment where a single paddock may cover several hundred square kilometres. Cattle do not strongly associate with one another and instead exhibit general gregariousness rather than tightly knit social groups. Cattle exhibit home range fidelity and in arid Australia some cattle may graze up to 9km from their watering source each day.[33-37]

The cattle population dataset was created based on known densities,[25] property records and aerial survey data. Two of the six leases in the study area have virtually no management of cattle and no paddock structure, with cattle tending to aggregate along the floodplain on these leases. Periodic, *ad hoc* mustering is practiced. The four other leases maintain a number of breeder herds that are placed in paddocks with permanent water. Mustering occurs once or twice a year between May and October,[25] when weaners are removed from breeder herds and collected into an age or sex cohort.

A total population of cattle in the study region was estimated by multiplying the area of cattle habitat by the average density of cattle (7 cattle per sq. km). The area around water sources was assumed to be habitable and divided into 4 concentric rings to allow declining cattle densities as the distance from water

[a] Aerial Survey Logger. S. McLeod and J. Tracey, Vertebrate Pest Research Unit, NSW Department of Primary Industries, Orange

increases (see behaviour and management section for justification). These rings were 0–2, 2.1–4, 4.1–6 and 6.1–7.5 km from water. Densities of cattle for each ring were calculated using the arbitrary function $density = 14 - 2^{\frac{547x}{1000}}$ (where *x* is distance to water) in order to simulate a population that declines in density as distance to water increases. This resulted in densities of 13, 11, 7 and 1 cattle per sq. km for each ring, respectively.

Feral pigs are highly social animals that live predominantly in herds and in close proximity to water sources.[28,38,27,39–42] Previously, Cowled et al.[40] used information from the literature to develop a feral pig distribution in the study area. In the current study this distribution was modified based on results of the aerial surveys conducted. This included modified estimates of the overall density of feral pigs in the study area, and the maximum distance pigs were observed from major waterways.

A stochastic spatially explicit micro-simulation model that operates within a GIS was developed. The model was adapted from an approach previously described for modelling CSF incursions in feral pigs in Australia.[40] A state-transition approach is used to represent the infection process and herds (pigs or cattle) may transition through Susceptible–Latent–Infectious–Recovered states. The application was coded in MapBasic®, and implemented in Mapinfo® (available from Pitney Bowes, http://www.mapinfo.com/products/applications/mapping-and-analyticalapplications).

The model takes into account spatial relationships, cattle and feral pig social structures and species ecology and behaviours, including management practices in the case of cattle. Social units (herds) of pigs and cattle are represented individually and all units have an area over which they will move each day (daily home range)

Within the model, pig–to–pig transmission can occur when daily home ranges of infectious and susceptible groups intersect, and the daily probability of infection was assumed to be 0.268. Cattle–to–cattle transmission may occur through the following infection pathways:

1. Shared watering points – assumes that all herds within the same paddock that share a watering point with an infected herd have a daily probability of infection.
2. Proximity – herds within the same paddock that do not share a watering point may come in 'contact' (as measured by intersecting daily home ranges) also have a probability of infection.
3. Indirect contact – fomite transmission between cattle herds on the same lease associated with normal management practices.
4. Cattle movements – seasonal movements that mix and move cattle (for example, turn- off, weaning).

The daily probability of infection for these pathways were assumed to be 0.138, 0.049, 0.0014 and 4.75, respectively, based on previous research and expert opinion.[43,44]

Transmission of infection from infectious pig herds to susceptible cattle herds, and from infectious cattle herds to susceptible pig herds, may occur when daily home ranges intersect, proportional to area of intersection, time since the source herd was infected (within-herd prevalence) and size of the source and exposed herds. The risk of infection is higher from pigs to cattle than from cattle to pigs since pigs excrete larger amounts of virus than cattle, and cattle are highly susceptible to infection by inhalation compared to pigs. The assumed daily probability of infection was 0.134 and 0.098, respectively.

Periods of cattle and pig herd latency, infectiousness and immunity were modelled as triangular statistical distributions, as previously described by Ward et al.,[43] based on expert opinion.

The model simulates control measures consistent with Australia's veterinary emergency control plan (AUSVETPLAN) for FMD.[45] This involves quarantine of infected premises and area movement restrictions, tracing of animal movements and surveillance, and culling of infected and exposed animals on infected and dangerous contact premises. Vaccination is unlikely to be considered in this remote area with low stocking rates. In the event that wild animals are found to be infected a wildlife population reduction program would be applied.[46]

Disease control in cattle involves three measures: all direct cattle movements and indirect contact cease after the index case is discovered, surveillance through stock inspections is implemented, and stamping out (destruction and disposal of cattle on infected premises) is carried out. We assumed all infected cattle herds would be detected. The time from onset of clinical signs to reporting was modelled as a triangular statistical distribution (7, 10, 14 days). We assumed that it would take one day to muster and cull cattle in an infected paddock. Because of the very large size of pastoral holdings in the study region we assumed that only cattle in paddocks where infection is found would be destocked. Disease control in feral pigs is based on a control zone of 10 km radius around infected herds that have been detected. Within this zone pig herds are culled, based on the likelihood that individual groups are sighted and culled. We assumed the probability of sighting and culling pig herds in the control zone to be 80%.[47] A 10 km surveillance zone is applied outside the control zone and sampling of herds in the surveillance zone is also undertaken. If new infected cattle or pig herds are detected then control and surveillance activities are expanded appropriately.

We consider initial infection of feral pigs with subsequent transmission within the pig population and between the pig and cattle populations (mixed species infection) to be the most plausible way FMD would be introduced and spread in this region and accordingly this is our *reference scenario*. To investigate the importance of the multi-host system, the potential spread of disease in pig populations only and in cattle populations only (single species infection) were

simulated separately. To start a simulation FMD virus was introduced to a randomly selected feral pig or cattle herd. Infection was then allowed to spread for 6 months with no control implemented. Two hundred simulations were used for all scenarios.

Given the uncertainty around FMD transmission in this setting – FMD has never occurred in this region – a sensitivity analysis of the transmission probabilities was undertaken by halving and doubling the baseline parameter estimates.

In a second set of studies, the effectiveness of control measures was evaluated in a mixed species outbreak using the *reference scenario* (FMD randomly introduced into the pig population with transmission from pig to cattle and cattle to pigs permitted). Disease was assumed to be detected 30, 60 or 90 days after introduction. The effects of targeted control of pigs only, cattle only and pigs plus cattle were separately considered. Again, the model was run for 6 months with 200 simulation runs in each case.

The mean proportion of FMD introductions in which disease established (still spreading at 6 months), the mean number of infected herds at 6 months, the mean incidence rate (number of herds infected per day), the mean total area infected (sq. km) at 6 months and the mean cumulative incidence were calculated for both feral pigs and cattle.

4. Results

Cattle sampling and analysis

Between 28 May and 1 June, 2011 a total of 496 fecal samples were collected from extensive beef cattle grazing on 3 pastoral leases in the Fitzroy Crossing district (Go Go Station, 346 – 70%; Jubilee Downs, 70 – 14%; Quanbun 80 – 16%).

Full access to the leases was available, as well as the history of livestock management in paddocks of interest. A simple random cell selection design was used across the study area to sample cattle. This ensured randomized selection of cattle and unbiased inferences. A helicopter was used and was the most economical and practical means of sampling to ensure the collection of fresh faeces.

Samples were collected from 32 geocoded mobs (median sample size 13, range 1-32) in 27 paddocks (**Figure 1**). The majority of cattle sampled were adults (84%). Some steers (60) and weaners (20) were also sampled. The average weight of cattle in mobs sampled was estimated to be 288 kg. Cattle sampled were on billabongs (51), bores (110), creeks (125) or dams (120), or were not near water sources when sampled (100) (**Figure 2**).

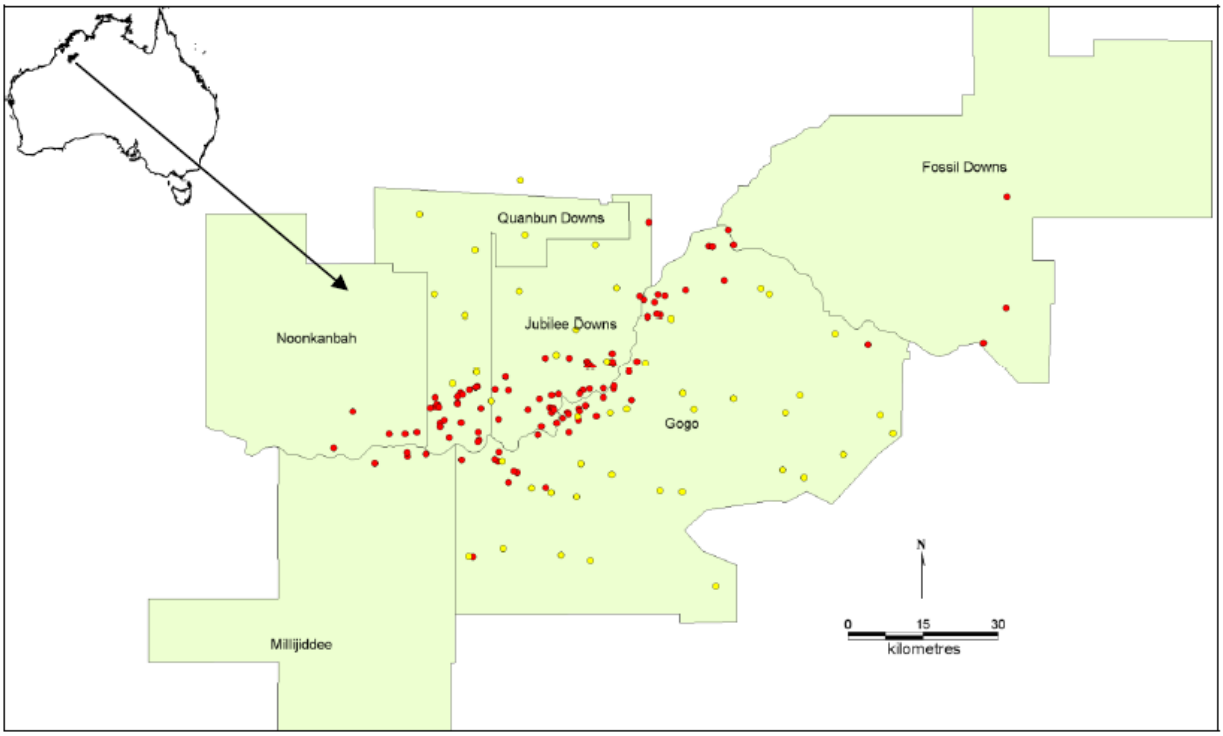


Figure 1: Location of study site and sampling locations in the Kimberley region of Western Australia. The yellow dots represent cattle herd sampling locations, whilst the red dots represent previously sampled feral pig herds.

Fecal samples were transported to Elizabeth MacArthur Agricultural Institute within 72 hours and cultured for *Salmonella* using standard microbiological techniques. Confirmed colonies of *Salmonella* were then forwarded to the National Reference Laboratory, Adelaide, for serotyping. Genotype analysis (pulsed-field gel electrophoresis) was undertaken.

Salmonella was isolated from 10 samples (2.02%; 95% CI, 1.03–3.80). From one sample, two different serotypes of *Salmonella* (Bukavu and Chester) were identified. S Chester was isolated from 3 samples. The serovars isolated in this study are shown in **Table 1**.

Table 1: Salmonella serovar isolated from 496 grazing cattle in the Kimberley region of northwestern Australia.

***Salmonella* serovar**	**Number isolated**
Bukavu	1
Chester (3)	3
Montevideo	1
Orion	1
Reading	1
Rubislaw	1
Treforest	1
Urbana	1
Wandsworth	1

Salmonella was isolated from GoGo (5; 1.45%), Quanbun (3; 3.75%) and Jubilee (2; 2.86%) leases (**Figure 2**).

Salmonella was isolated from two samples each from two mobs (prevalence 20%). In the remaining positive mobs, prevalence ranged from 5 to 10%.

Samples collected from mobs on bores or dams (9/221; 4.07%) were more likely to be *Salmonella* positive than those from mobs on billabongs and creeks (1/165; 0.61%) or not on a water source (0/100), P = 0.0383.

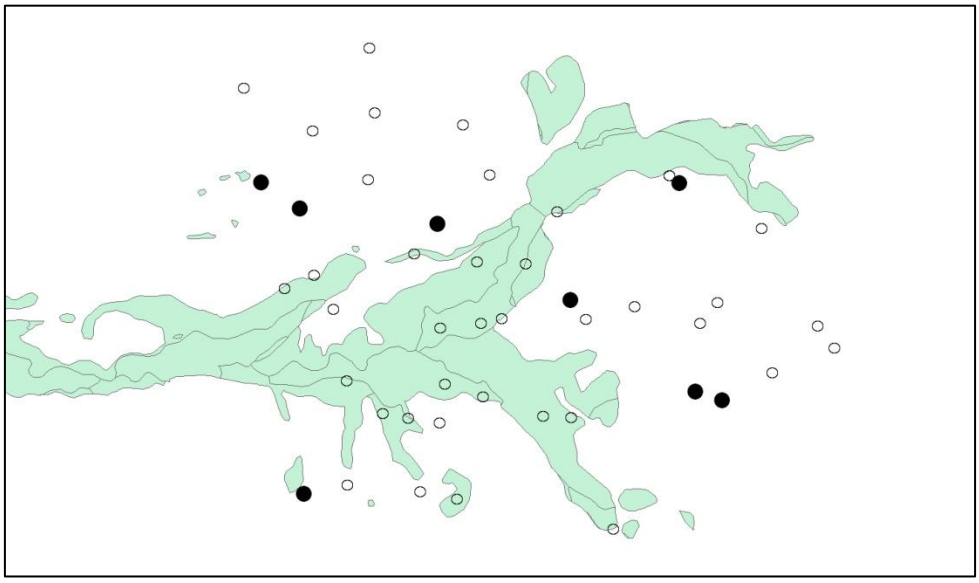


Figure 2: Location of cattle mobs sampled for *Salmonella*, Fitzroy River district (open circles) and locations at which *Salmonella* was detected (closed circles).

Of the 9 different serotypes isolated from cattle, 6 of the same serotypes were isolated from feral pigs (number of pigs), either from fecal samples or mesenteric lymph nodes: Chester (2), Montevideo (6), Orion (1), Rubislaw (2), Urbana (1) and Wandsworth (1). No spatial overlap between *Salmonella* positive cattle mobs and feral pigs locations at which these same serotypes were isolated was apparent (**Figure 3**).

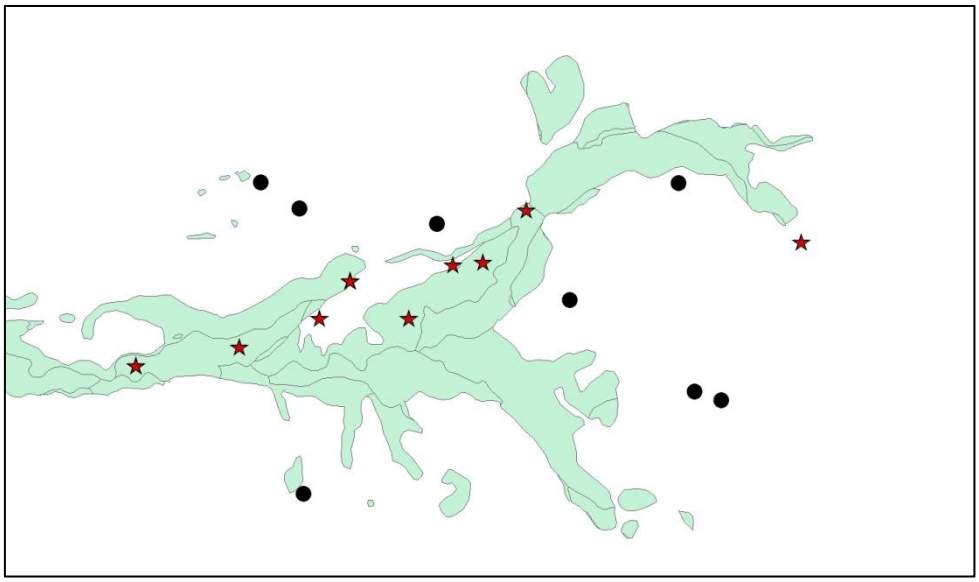


Figure 3: Location of cattle mobs, Fitzroy River district, from which *Salmonella* was isolated (closed circles) and locations at which the same *Salmonella* serotypes (Chester, Montevideo, Orion, Rubislaw, Urbana and Wandsworth) were isolated from feral pigs (stars).

Table 2 shows a comparison of the 6 serotyes isolated from cattle in common with pig isolates: Chester (2 pig isolates), Montevideo (6), Orion (1), Rubislaw (2), Urbana (1) and Wandsworth (1).

Cattle serotypes were matched with corresponding pig serotypes and genetic similarity was determined, based on pulsed field electrophoresis (PFGE) analysis (**Table 2**). The average similarity was 76.51% (95% CI, 73.65−79.37), compared to the average similarity of all cattle and pig serotypes (n = 299) of 51.98%. However, when compared to the average similarity for these specific serovars isolated from pigs only (**Table 3**), 4 of the serovars (Salmonella Chester, Montevideo, Urbana and Wandsworth) were more similar (range 4.12−13.34%) in the pig−pig comparison than the cattle−pig comparison. For only two serovars (*Salmonella* Orion and Rubislaw) were the pig−pig comparisons less similar (2.27 and 3.88%) than the cattle−pig comparisons.

Overall, no evidence was found to suggest direct transmission of *Salmonella* between feral pigs and cattle co-grazing in the study area.

Table 2: Comparisons between cattle and feral pig isolates of *Salmonella* of the same serotypes, showing genetic similarity measured by pulsed field electrophoresis (PFGE).

Cattle		Pig			
ID	**Serotype**	**ID**	**Serotype**	**Tissue**	**Similarity**
28	Chester	39	Chester	Faecal	76.93
28	Chester	40	Chester	MLN	83.33
28	Chester	175	Chester	Faecal	88.89
32	Chester	39	Chester	Faecal	85.72
32	Chester	40	Chester	MLN	76.93
32	Chester	175	Chester	Faecal	81.49
384	Chester	39	Chester	Faecal	76.93
384	Chester	40	Chester	MLN	80.01
384	Chester	175	Chester	Faecal	74.08
440	Montevideo	72	Montevideo	Faecal	83.33
440	Montevideo	73	Montevideo	Faecal	81.49
440	Montevideo	74	Montevideo	Faecal	81.49
440	Montevideo	75	Montevideo	Faecal	75.00
440	Montevideo	76	Montevideo	Faecal	81.49
440	Montevideo	77	Montevideo	Faecal	81.49
440	Montevideo	216	Montevideo	Faecal	88.89
440	Montevideo	217	Montevideo	Faecal	88.89
440	Montevideo	218	Montevideo	Faecal	81.49
440	Montevideo	277	Montevideo	Faecal	84.62
440	Montevideo	278	Montevideo	Faecal	84.62
398	Orion	259	Orion	Faecal	86.96
398	Orion	275	Orion	Faecal	58.33
437	Rubislaw	251	Rubislaw	Faecal	78.26
15	Urbana	128	Urbana	MLN	69.23
15	Urbana	165	Urbana	Faecal	59.26
15	Urbana	191	Urbana	Faecal	59.26
15	Urbana	226	Urbana	Faecal	83.33
15	Urbana	246	Urbana	Faecal	76.93
15	Urbana	257	Urbana	Faecal	75.00
15	Urbana	268	Urbana	Faecal	75.00
231	Wandsworth	129	Wandsworth	Faecal	72.73
231	Wandsworth	135	Wandsworth	Faecal	83.33
231	Wandsworth	190	Wandsworth	Faecal	66.67
231	Wandsworth	265	Wandsworth	Faecal	73.33
231	Wandsworth	276	Wandsworth	Faecal	66.67

Table 3: Comparisons between cattle−pig and pig−pig isolates of *Salmonella* of the same serotypes. Average genetic similarity measured by pulsed field electrophoresis (PFGE) is shown for each serotype for both comparison type.

Serovar	cattle – pig	pig – pig	Difference
Chester	80.48 ↓	84.60	− 4.12
Montevideo	82.98 ↓	96.32	− 13.34
Orion	72.65 ↑	68.77	+ 3.88
Rubislaw	78.26 ↑	75.99	+ 2.27
Urbana	71.14 ↓	80.52	− 9.38
Wandsworth	72.55 ↓	81.42	− 8.87

Feral pig sampling and analysis

Feral pig sampling was completed in October 2010 and feral pig genotyping was completed in October 2011. In total 543 feral pig genotypes were analysed across 14 loci. The median pairwise pig genetic dissimilarity was 39 (IQR: 35–46, range: 7–71). The sampled population was remarkably genetically homogenous.[28]

Disease spread modelling

During aerial surveys, 1263 cattle herds were observed. The median herd size was 4 cattle (Q1–Q3: 2–10) with a range of 1 to approximately 1000 cattle. Cattle herd sizes observed during aerial surveys resembled a Poisson or negative binomial distribution with a mean of 1, although over dispersion was also evident. Cattle herd sizes were arbitrarily simulated using a Poisson distribution (20% of herds, mean=1), a uniform distribution (0.5% of herds 1–1000) and a BetaPert distribution (79.5% of herds, lowest=1, most likely=3, highest=40) to derive a probability distribution that visually resembled that observed during the aerial survey. These herds were then randomly distributed across the study area, although care was taken to distribute them in concentric rings around waterways according to the densities derived above. The study area was estimated to contain approximately 79,400 cattle in 8,231 functional herds in 84 paddocks covering the 6 pastoral leases.

Overall, a total of 208 feral pigs in 48 groups were counted in the aerial survey, an estimated density of 0.62–1.68 feral pigs per sq. km. Assuming 1 pig per sq. km of suitable habitat for the entire aerial survey area (6,818 sq. km), 1190 pigs located in 275 functional herds was used in disease spread modelling.

In the mixed species infections, outbreaks of FMD were predicted to establish and still be spreading at 6 months in 75–81 % of introductions, with larger outbreaks being seen when disease was introduced via the pig population

(*reference scenario*) (**Table 4**). A typical example of a disease outbreak simulation is shown in **Figure 4**. Smaller outbreaks were seen in the single species scenarios. In the cattle only scenario, FMD was also likely to establish and spread, with infection still active at 6 months in 62% or runs. In contrast, in the pig only scenario, FMD inevitably died out without intervention. The median survival time was only 19 days (95% prediction interval, 12–52). In 64.5% of runs infection did not spread beyond the initial infected herd, compared to 4.5% of runs in the cattle only scenario.

The largest outbreaks in both cattle and pigs (herds infected and cumulative incidence) and area infected occurred in the *reference scenario* (**Table 4)**: a median of 2941 (95% PI, 1–5658) cattle herds were predicted to be infected across an area of 5634 sq. km (95% PI, 0–9259). Cumulative incidence of infection for cattle herds was 35.7% (95% PI, 0–72.9). In addition, there was a median of 87 (95% PI, 1-186) pig herds infected with a cumulative incidence of 31.5% (95% PI, 0.3-67.6). Mixed species infection initiated in the cattle population was slightly smaller. For cattle only outbreaks, fewer herds were infected: median cumulative incidence 14.9% (95% PI, 0-60.7). In contrast to the mixed and cattle only scenarios, for the pig only outbreaks there was very little spread: median cumulative incidence 0.4% (95% PI, 0.4-1.5).

Not surprisingly, increasing the value of the epidemiological transmission parameters for the *reference scenario* resulted in a greater proportion of epidemics establishing—91% compared to 81% for the baseline parameters (**Table 5**). It also resulted in a larger epidemic size, particularly for the predicted number of herds infected and size of area infected (1.5–2.1 fold increases). Conversely, decreasing transmission parameters reduced the proportion of epidemics that established and spread (57%), and reduced the number of infected herds and size of the area infected (**Table 5**).

A control strategy targeting feral pigs only was not predicted to be successful. Assuming FMD was detected 30 days after introduction, in a control program focused only on feral pigs, but involving cattle, 39% of outbreaks would still be active at 6 months (**Table 6**). A control program focused on cattle only, or including both cattle and pigs, always resulted in eradication within 6 months. Compared to control targeting pigs only, targeting both pigs and cattle resulted in larger control areas, a similar number of pig herds culled but a greater likelihood of eradicating the disease (100% compared to 61%) with an average 69-day reduction in the time needed to control an outbreak (**Table 6**). Compared to control targeting cattle only, targeting both pigs and cattle resulted in shorter outbreaks (on average a 7-day reduction), fewer cattle herds culled and a smaller control area (**Table 6**). Even with delayed detection (at 60 days and 90 days), a control strategy in which both cattle and pigs were targeted minimised the time to eradication and number of cattle culled (data not shown).

Table 4: Predictions from a model simulating infection of extensively managed cattle and feral pigs by FMD virus in north-west Australia, based on different assumptions regarding intra- and inter-species disease transmission. No disease control was assumed and the model was simulated for 200 iterations for 180 days for each experiment.

		Cattle			Pigs		
Experiment	Outbreaks*	Herds infected†	Area infected‡	Cumulative incidence§	Herds infected	Area infected	Cumulative incidence
Pig–to–cattle‖	81	2941 (0-6000)#	5634 (0-9259)	35.7 (0-72.9)	87 (1-186)	3205 (0-5634)	31.5 (0.3-67.6)
Cattle–to–pig	75	2373 (1-5658)	4645 (0-8724)	28.9 (0.1-68.7)	79 (0-170)	2506 (0-5501)	28.5 (0-61.8)
Cattle–to–cattle	62	1223 (1-5001)	2585 (0-7945)	14.9 (0-60.7)	–	–	–
Pig–to–pig	0	–	–	–	1 (1-4)	0 (0-3)	0.4 (0.4-1.5)

* proportion (%) of all simulations in which a single point introduction leads to disease transmission still occurring at 6 months
† total number of herds infected throughout the simulation
‡ area (sq. km) of a minimum convex hull (MCH) established around every infected herd throughout the epidemic. NB a MCH requires at least three points.
§ proportion (%) of herds infected, the total number of infected herds ÷ total herds in contiguous population
‖ reference scenario
median (95% prediction interval)

Table 5: Predictions from a model simulating infection of extensively managed cattle and feral pigs by FMD virus in north-west Australia, based on different assumptions regarding intra- and inter-species disease transmission. Sensitivity analysis of the reference scenario (FMD introduction in a randomly selected pig herd, transmission between pig herds, pig herds to cattle herds and between cattle herds; and no disease control) was performed by halving and doubling the baseline transmission.

		Cattle			Pigs		
Experiment	Outbreaks*	Herds infected†	Area infected‡	Cumulative incidence§	Herds infected	Area infected	Cumulative incidence
Baseline transmission	81	2941 (0-6000)ǁ	5634 (0-9259)	35.7 (0-72.9)	87 (1-186)	3205 (0-5634)	31.5 (0.4-67.6)
Half transmission	57	626 (0-4093)	1055 (0-6662)	7.6 (0-49.7)	12 (1-118)	263 (0-4218)	4.3 (0.4-42.7)
Double transmission	91	5902 (0-7419)	8469 (0-10469)	71.7 (0-90.1)	183 (1-222)	5098 (0-6923)	66.5 (0.4-80.7)

* proportion (%) of all simulations in which a single point introduction leads to disease transmission still occurring at 6 months

† total number of herds infected throughout the simulation

‡ area (sq. km) of a minimum convex hull (MCH) established around every infected herd throughout the epidemic. NB a MCH requires at least three points.

§ proportion (%) of herds infected, the total number of infected herds ÷ total herds in contiguous population

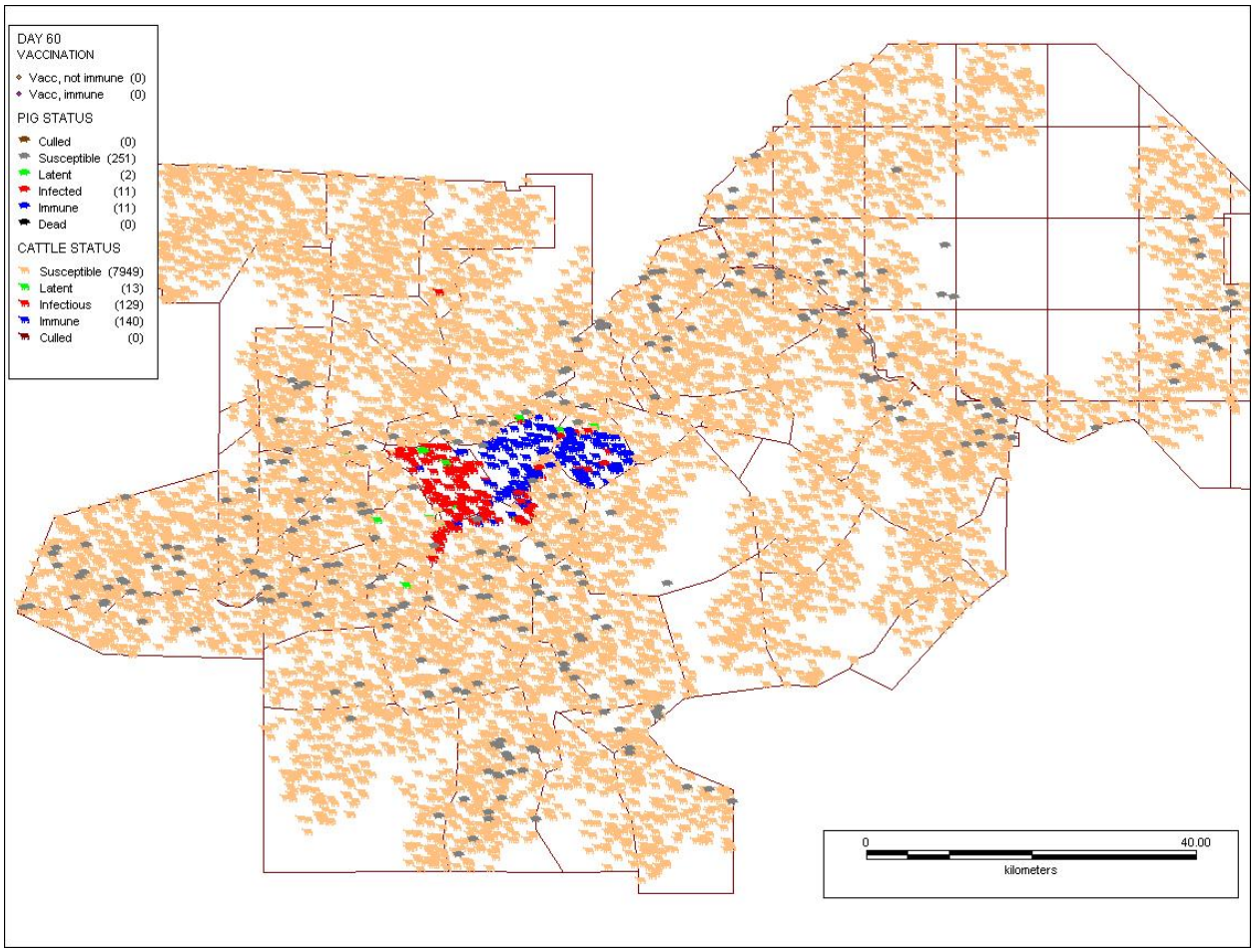
ǁ median (95% prediction interval)

Table 6: Predictions from a model simulating infection of extensively managed cattle and feral pigs by FMD virus in north-west Australia, using three different disease control strategies: culling pigs only, cattle only or cattle and pigs using the reference scenario (FMD introduction in a randomly selected pig herd, transmission between pig herds, pig herds to cattle herds and between cattle herds). The model was simulated for 200 iterations for 180 days for each scenario, and disease detection was assumed to occur at day 30. Note that disease died out before 30 days (without any intervention) in 21 out of 200 runs.

Metric	Control strategy		
	Pigs only	Cattle only	Cattle and pigs
Probability of eradication*	60.9%	100%	100%
Time to eradication (days)†	111 (52–174)	49 (31–105)	42 (31–74)
Control area (sq km)‡	146 (6–359)	335 (60–1079)	283 (55–1021)
Cattle herds culled	–	349 (83–941	329 (71–902)
Total cattle culled	–	3332 (730–9986)	3264 (730–8529)
Pig herds culled	20 (3–49)	–	20 (0–71)

* when eradication was achieved
† days until outbreak controlled: how many days from introduction until there are no infected cattle or pigs left (or if infection still present at 180 days, outbreak uncontrolled)
‡ area (sq. km) of a minimum convex hull established around every infected herd throughout the epidemic

Figure 4: An example of a disease outbreak simulation of an FMD incursion in a population of extensively managed cattle and feral pigs in north-west Australia.



5. Discussion/conclusion

Cattle *Salmonella* prevalence within the study area of Fitzroy Crossing was found to be much lower than the feral pig prevalence (~38%). In addition, cattle *Salmonella* isolates were more commonly from areas without feral pigs, but conversely, from high density cattle populations on artificial water sources. No obvious associations were identified between the cattle serotypes and those isolated from the co-grazing feral pig population in this study area. Pulsed-field gel electrophoresis analysis confirmed this observation.

The feral pig population in the study area was found to be remarkably homogenous, and unlike other pig populations studied in Australia (for example see Hampton et al.[21] and Cowled et al.[19]). In addition, aerial surveys revealed that its density was lower than previously suspected, and that the spatial distribution was disjointed, focused on areas of water and rich in natural resources. This has implications for our understanding of how an incursion of FMD might behave in such an environment.

Based on field studies of the interaction between domestic cattle and feral pigs in the Kimberley region, the potential spread and control options for an FMD incursion in northern Australia were investigated, using a disease spread model. Depopulation of feral pigs only was not predicted to be successful. Movement standstill, surveillance and depopulation of cattle only would successfully eradicate the disease. However, control targeting both feral pigs and cattle would result in smaller outbreaks. If FMD is controlled in cattle, then it is likely to be self-limiting in feral pigs. To eradicate an FMD incursion as quickly as possible, both feral pigs and cattle should be targeted.

This study has demonstrated that a different disease pattern will occur in a two-species disease ecosystem than will be seen if each species is considered in isolation. Considering feral pigs in isolation, FMD inevitably died out in a relative short time frame (weeks) in the Kimberley environment. This is not surprising given the relatively small number and limited distribution of pig herds (based on aerial surveys, we estimated that there were 1,190 pigs in the study area) compared to cattle (approximately 79,400 grazing cattle). In cattle only, the disease was more likely to establish and spread, although there was a 38% probability that it would die out within 6 months. However, when feral pig-cattle interactions were taken into account, outbreaks were invariably larger and disease more likely to persist. The findings suggest that if FMD is controlled in cattle, it is likely to be self-limiting in feral pigs. This has important implications in terms of disease response and resource management in this remote region.

In the event of an FMD incursion in this wildlife-livestock ecosystem, simulation results suggest that it is the cattle population that determines the outcome. The likely reasons for this include that cattle in the study area exist at higher densities, are more dispersed, have larger home ranges (because this species is

less reliant on watercourses) than the feral pig population and are moved large distances during routine management practices. Because of the inter-connectiveness of the cattle population, disease could be sustained, thus allowing regular spillover of infection to feral pigs that share this landscape.
Disease control focusing on depopulation of feral pigs was predicted to lead to only slightly smaller outbreaks compared to the uncontrolled situation (*reference scenario*). There was only a 61% probability of FMD being eradicated with a pig-only control program. In contrast, control only in cattle always leads to eradication, suggesting that pigs at the density observed in our study area would act as a spillover species for FMD. However, control of both pigs and cattle resulted in the shortest time to eradication. Thus, if time to eradication is a driving force in the response to an incursion (that is, to regain FMD-free status and resume trade) then both species need to be included in the disease management plan. If there are limited resources available, then focusing on controlling FMD in cattle is likely to be the preferred approach in the first instance. Once this has been achieved, then assessing the disease status of the feral pig population would become a priority.[48] Although the role of feral pigs in this ecosystem in the spread of disease might be minor, an equally important issue is the demonstration of disease freedom once an incursion has been control.

Previous disease spread modelling of CSF in this population of feral pigs found that disease was likely to spread quickly.[40] However, in that study higher feral pigs densities (based on expert opinion) were assumed. In the current study we used lower density values based on an aerial survey. The different findings in the two studies suggest that the optimal approach to managing an exotic disease incursion involving feral pigs is likely to be very sensitive to the distribution and density of the pig population. If this is the case, then a key component of the response should be to determine the distribution and abundance of the local feral pig population (also recognising that wildlife populations can change quickly in response to weather and other seasonal events and the availability of natural resources). Based on distribution and abundance estimates, an optimal response strategy can then be developed. It should be noted that the response will also depend on the nature of the disease. In the case of CSF, a persistent carrier state in pigs exist, but this is not the case for FMD. This also needs to be taken into account when assessing the role a species may play in maintaining and spreading disease.

The sensitivity analysis of the simulation model used in this study identified (as expected) the critical importance of understanding within- and between-species transmission. We assumed that the daily probability of infection occurring, given that two feral pig herds (infectious and susceptible) come into contact, was 0.268. It should be noted that the actual parameter value is weighted by within group prevalence of the infectious group, so that the actual average daily value was 0.103 in our simulations. In essence, if two pig herds' home ranges intersect on any given day the average daily probability that transmission would occur is about 10%. This value will vary with the stage of infection in the infectious herd.

Also, transmission based on the intersection of home range assumes both direct and indirect contact, that is, the temporal component of actual daily movement within a herd's home range is ignored. Retrospective analysis of model output of pig only runs (n = 50) indicated that, based on the number of newly infected herds during the simulations, the daily transmission rate was 0.026. Assuming an average 14-day infectious period, this would equate to an interherd basic reproduction number (R_O) of 0.36 (95% CI, 0.23–0.5). Thus, it is not surprising that most simulated epidemics in the pig only transmission scenario died out. Even doubling the pig-to-pig transmission probability had little effect (results not shown) suggesting that it is a lack of contact between herds in the study area that is important. In fact, the estimated average daily contact rate in our dynamic model (for each simulation, the total contacts that infectious groups had divided by the duration of the outbreak) was 0.225 per day. An infectious herd would have an average of 3 contacts over its infectious period in this environment, with only about a 10% chance that transmission would occur (assuming the contacted group is susceptible).

For cattle, retrospective analysis of model output indicated that the daily transmission rate was 0.212 and assuming an average infectious period of 17 days, the estimated R_O was 3.6 (95% CI 3.1 – 4.1). This value is plausible; for example, Perez et al.[49] estimated that the interherd R_O for the 2001 Argentine FMD outbreak in cattle ranged from 2.4 to 3.8, prior to implementation of control measures. In contrast, we estimate R_O for FMD transmission between domestic pig herds in Taiwan in 1997[50] (based on 717 herds infected during the first two weeks prior to implementation of mass vaccination and an average herd infectious period of 14 days) to be approximately 2.0. This illustrates the unique characteristics of the ecosystem simulated in the current study – the grazing cattle population are likely typical of extensively managed systems throughout the world, whereas disease transmission in the feral pig population is very different from the situation in domestic pig production systems. When these two species are considered within the same wildlife-livestock ecosystem, the spread of FMD predicted by disease modelling can reveal insights that inform disease control policy.

The current AUSVETPLAN for FMD states that 'Destruction of infected and suspect infected animals should be completed as rapidly as possible to reduce shedding of the virus and spread of disease.' Vaccination may be considered in some circumstances, but not as a substitute for movement controls and other biosecurity measures. It is unlikely that vaccination would be used in the Kimberley region, since vaccinated stock would be ineligible for export.

Within the studied ecosystem, the ability of each species to spread and sustain FMD is likely different. Due to management (cattle maintained at much higher density), ecology (cattle can roam further from water sources) and epidemiology (cattle might act as the disease reservoir in this ecosystem), control of the disease in cattle needs to be prioritised. However, to quickly achieve eradication

and importantly, to demonstrate disease freedom, response strategies must include feral pigs. The results of this simulation study puts into perspective the role that feral pigs might play in an incursion of FMD. The eradication of feral pigs will not substantially reduce the risk of FMD outbreaks in such northern Australian ecosystems, but the control of feral pigs remains important after an outbreak occurs.

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